Wound Botulism Resulting from Heroin Abuse: Can You Recognize It?

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CE Earn Up to 9 CE Hours. See page 312.

A 46-year-old woman presents to triage because she has had trouble speaking and swallowing since she awoke that morning. She is alert and oriented, but her speech is a slightly garbled. Her vital signs are as follows: heart rate, 86 beats per minute; respirations, 16 per minute; blood pressure, 136/86 mm Hg; temperature, 37.2°C (98.9°F); and oxygen saturation, 96% on room air. Her medical history is negative except for hepatitis C and frequent urinary tract infections. Her social history is positive for smoking one pack per day, as well as abuse of prescription narcotic medications and, more recently, heroin. She tells you the last time she shot up was 3 days ago.

How would you triage this patient?

The Problem

Classically, botulism toxicity is associated with improperly canned foods or ingestion of honey by an infant. Recently there has been an increased incidence of wound botulism associated with intramuscular or subcutaneous injection of street drugs, particularly heroin. The purpose of this article is to raise awareness of this problem and report its typical presentation so emergency nurses can provide an effective rapid response to this emergent condition.

Botulism Overview and Clinical Presentation

Botulism is a disease caused by the toxin of *Clostridium botulinum*, an anaerobic gram-positive bacterium common to soil worldwide. *C. botulinum* produces a highly potent neurotoxin and spores that can survive in extreme environmental conditions.[1-3] When humans are infected, the toxin enters the bloodstream and paralyzes muscles by irreversibly blocking the release of acetylcholine at peripheral neuromuscular junctions. The hallmark features of botulism are cranial nerve palsies followed by symmetric descending paralysis. Many patients who present to the emergency department report having trouble swallowing, blurred vision, slurred speech, and weakness of the face and limb muscles.[3] Indeed, speech and visual disturbances characterize botulism's early signs, which include dysarthria, dysphonia, and poor accommodation often with mydriasis. Weakness is mild and descending, but sensation and mentation are unaffected. Fever is absent unless an inflamed skin abscess is present with a secondary infection.[4] As paralysis progresses, symptoms worsen over several hours with much more marked weakness, ophthalmoplegia, ptosis, dysphagia, speech difficulties, dyspnea, and possibly respiratory failure requiring mechanical ventilation.

Most cases of botulism toxicity are 1 of 3 main types: food botulism, infant botulism, and wound botulism.[1,2] Food botulism is caused by the ingestion of the toxin, usually from improperly home-canned foods. In food botulism, the hallmark neurologic symptoms are accompanied by symptoms of gastroenteritis. Infant botulism is caused by the ingestion of spores, particularly those found in honey, that the immature digestive tract is unable to inhibit.[1] Historically, infant botulism and food botulism have accounted for most of the 110 average annual cases in the United States, with the Centers for Disease Control and Prevention reporting 72% of cases being infant botulism and 25% being food botulism.[1] However, cases of wound botulism have been increasing recently, which until the early 1990s had resulted predominately from traumatic injuries or contaminated surgical wounds.[1-3] The recent rise of wound botulism presents mainly in people who inject heroin, and sometimes cocaine, subcutaneously or intramuscularly.[1-3]

Injection Drug Use: An Opportunity for Wound Botulism

Most heroin users inject intravenously, but if intravenous access is difficult to obtain or personal preference dictates,

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the user may inject subcutaneously ("skin popping") or intramuscularly ("muscling"). With intravenous injection, the aerobic environment does not allow proliferation of botulinum toxin. Skin popping or muscling, however, creates a favorable anaerobic environment for botulinum spores to germinate because repeated injections and the solvent or drug's diluents may cause local tissue necrosis[3,5-7] When heroin is injected parenterally it is first dissolved in a solvent such as water, lemon juice, or another weak acid, heated, and filtered through a cotton ball or cigarette filter before being drawn up into the syringe. At an unknown point in its processing it becomes contaminated with *C. botulinum* spores, possibly at one of the steps when it is "cut" or diluted with one of many dilatants such as sugar, strychnine, Xylocaine, dirt, or paper soaked with shoe polish. The process to prepare the drug for injection may kill competitive bacteria and promote the germination of the *C. botulinum* spores.[3,5,8,9] The spores germinate into bacteria that produce toxin. It is difficult to ascertain the incubation period between injection and onset of symptoms because heroin users tend to inject frequently.[3]

Demographics

Patients with wound botulism from heroin injection tend to be in their 20s to 50s. While women only represent about one quarter of all heroin users, 30% to 50% of patients with wound botulism are women, perhaps because they are less likely to inject intravenously than are men. More are white or Hispanic than are Asian or African American.[3] Most cases of wound botulism have occurred in the United States, and the vast majority of those have occurred in California; these cases are believed to have been caused by contaminated "black-tar" heroin imported from Mexico.[7,10] Since the early 2000s an upsurge of cases has occurred in the United Kingdom and other parts of Western Europe, where heroin is generally imported in a powdered form from Asia.[5,9,11,12]

Identifying Wound Botulism

When a patient presents with a history of heroin use and symptoms of cranial nerve paralysis, suspicion should remain high for wound botulism. The presentation of ptosis, weakness, and dysarthria can mislead the health care provider into thinking the patient is intoxicated, particularly if the patient is known to abuse alcohol or street drugs. If symptoms have not progressed sufficiently, the patient's potential to deteriorate may not be appreciated. A complete blood cell count, chemistry, urinalysis, cerebrospinal fluid, and liver function tests are all likely to be within normal limits unless a secondary infection is present.[2,3] These test results can further inappropriately minimize suspicion for a patient's potential to progress to respiratory failure. Assessment of the patient's ability to maintain his or her airway is paramount, especially when the patient reports having trouble swallowing or speaking. The nurse also should perform a thorough examination of the skin for evidence of injection, especially at unusual injection sites such as the thighs and buttocks. Any abscesses that might be a source of botulism toxin growth should be noted. These abscesses are easily missed because they can be small and minimally inflamed.[2,4,7] Peripheral intravenous access may be difficult to obtain, and thus central venous access may become necessary for obtaining blood specimens and administering fluids and drugs.

Differential diagnoses include a variant of Guillain-Barré syndrome, autoimmune neuromuscular diseases such as myasthenia gravis and Lambert-Eaton syndrome, organophosphate poisoning, paralytic shellfish poisoning, tick paralysis, or poliomyelitis.[2-4,7,13,14] Definitive diagnosis of botulism is identification of the neurotoxin in serum or wound aspirate.[1-3,5] This neurotoxin is identified in most but not all cases, and the earliest obtained serum specimen should be tested to decrease the likelihood of a false-negative result.[1,3,4] Electromyogram findings, while not always present, can be very helpful in arriving at a clinical diagnosis while awaiting serum toxin results.[1,3,4]

Treatment and Supportive Care

Early administration of botulism antitoxin is the most important treatment for a patient with wound botulism. It is most effective if given within 24 hours of the onset of symptoms because the antitoxin only acts on free neurotoxin that has not yet been taken up into the motor neuron and does not reverse the action of toxin that has already disabled acetylcholine release at a neuromuscular junction.[13] The decision to treat therefore should be made upon clinical suspicion of botulism, not on laboratory confirmation, because obtaining results may take several days.[2-4,13] Early administration of antitoxin decreases the length of ventilator support and time in critical care settings.[3,6,10] Because toxin continues to be produced within the anaerobic environment of the heroin injection site, surgical debridement of the wound also is recommended to stop disease progression.[2,10] Some but not all studies support the correlation of early debridement to shorter hospitalizations.[2,10]

Antibiotic therapies can be effective against *C. botulinum,* but their helpfulness or necessity has not been established. Aminoglycosides and clindamycin should be avoided because they can increase presynaptic neuromuscular block-

age.[2,3,13] Most other care is supportive. Not all patients require ventilatory support, but many do, and when tidal volume reaches 30% of expected, elective intubation should be executed.[3] Intubation also should be initiated if paralysis of the cranial nerves prevents the patient from maintaining his or her own airway. Patients may remain on a ventilator for weeks to months while new axon terminals are generated to replace the disabled ones.[2] Hydration and nutrition may need to be administered parenterally or via a nasogastric tube because swallowing is often affected. Patients often require extensive rehabilitation after prolonged convalescence to regain full function, but complete recovery is expected for most patients.[2,3] The survival rate from botulism is about 85% to 90% and is correlated with the expediency of intervention upon respiratory failure.[2,3]

The Emergency Nurse: A Vital Role

The presence of wound botulism in people who inject drugs intramuscularly or subcutaneously is a relatively new and increasing problem. A nurse's early recognition of the signs of botulism toxicity can be indispensable for diagnosis and intervention before progression to respiratory failure. Airway and ventilatory support remain the primary ED interventions. Anticipation of need for specimens and electromyography for diagnosis and administration of antitoxin and surgical debridement for treatment are key in the initial care of a patient with wound botulism.

You recognize that your patient may have wound botulism related to her heroin abuse. You bring her back to a room and alert the ED physician about your concerns. Upon testing, it is discovered that the patient does not have a gag reflex. She is intubated. Because she is unable to obtain intravenous access, the physician starts a central line and blood is obtained for testing of C. botulinum *toxin. Electromyography and neurosurgery specialties are not available at your facility, so your patient is prepared for transfer to a higher level of care. Electromyography later suggests a clinical diagnosis of botulism, so she is treated with the appropriate antitoxin within 16 hours of her initial presentation to your emergency department. Serum testing later confirms* C. botulinum. *The patient remains on the ventilator in the intensive care unit for 3 weeks before being discharged with speech therapy and outpatient drug rehabilitation.*

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